THE VETERINARY JOURNAL

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SEPTEMBER, 1944

EDITORIAL

VETERINARY PHYSIOLOGY

In a recent issue of The Veterinary Journal we drew our readers' attention to the paucity of clinical articles which appear in the veterinary periodicals, and pointed out that much of the experiences of clinicians was being lost to recent graduates by the failure to publish interesting observations. We welcome the contribution in this number of the "Observations on Equine Colic" from the pen of one who has given much thought to the subject and who has come to conclusions from his personal contact with the condition. In this article there are raised some doubts on some of the commonly practised treatments, with arguments on their usefulness. As with many of the diseases from which our domestic animals suffer, so is it with the group of diseases classed as "colic"—we shall not arrive at definite conclusions on their treatment until we have a better understanding of the normal physiological processes of the various systems. Two of the important systems—digestive and reproductive—are but imperfectly understood. The attention of our research workers has been largely given to the abnormal (disease) at the expense of the normal processes (health). It is gratifying to know that studies of some of the normal processes are now being made and that we may look forward in the near future to an extension of the work to include the processes and systems which have hitherto been neglected. It is only by a full knowledge and appreciation of the normal that we can hope to understand disease and its prevention and treatment.

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General Articles

OBSERVATIONS ON EQUINE COLIC

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THE word colic in the veterinary sense means literally pain in the intestine. For clinical purposes, however, spasmodic colic could be roughly defined as what we see in the behaviour of a horse when the normal gentle waves of intestinal peristalsis are thrown into a state of storm, breaking upon the rocks of a temporary or permanent intestinal stasis at a fixed point.

It is only within very recent times that it has become possible to imagine and explain the nature of spasmodic colic. I would like to make this point as clear as my limited knowledge permits, because by far the most helpful and comforting thing that can happen to a clinician in his work is to know exactly what he is up against. One begins by imagining the normal peristalsis with its mysterious and almost perpetual motion, so softly and evenly executed under normal conditions as to be imperceptible, so that in perfect health there is no consciousness of any movement except for purpose of defæcation. It used to be thought that peristalsis was due, like voluntary muscular reflex action, simply to local intestinal irritation sending afferent messages to the spinal cord or ganglia whence efferent messages ensured the requisite local muscular movement.

At least that is what I thought. But in perfect health there is no irritation and normal peristalsis must derive from some much finer and more sensitive mechanism. Modern biochemical work suggests that normal peristalsis is regulated by two or more ferments having an opposite action on the nerve terminals. One set, of which choline is an instance, is thought to stimulate and the other to depress the parasympathetic nervous system. Anything, then, which grossly excites or depresses or otherwise upsets the evenly balanced and normally automatic action of these opposing ferments can cause excessive spasm or excessive stasis of part of the bowel and consequently colic.

Coincidentally flatulence occurs, increasing the pain and the danger of the colic, through distension of the walls of stomach or intestine.

A great variety of causes can upset the balanced secretion of the ferments at the nerve-endings, thus giving rise to symptoms of colic. Indigestion, impaction, volvulus, strangulation and several other conditions can exert much the same effect and produce much the same symptoms in the early stages.

Herein lies the eternal difficulty of the clinician who—even after forty years of observing equine colic—may be as much at a loss as the latest graduate to know what is the cause and consequently what will be the outcome of a case of colic when the attack begins.

One might interpolate here that in our ordinary farm practice in Tasmania the cause is—in nine cases out of ten—indigestion, and often a temporary slight impaction, due to too big a feed of dry oaten chaff or to an ordinary feed of dry oaten chaff eaten too quickly, or in a state of fatigue, or after insufficient water. A case from such a cause will almost always recover spontaneously and death—when it occurs—is commonly due to severe impaction or ruptured

stomach. That—so far—has been the common experience of the essayist on the North West Coast, as verified when possible by P.M. examination. Too many working horses are kept, when at rest, in stables eating nothing but dry chaff and being watered either insufficiently or too infrequently.

Symptoms

The symptoms of spasmodic colic are well known, but there are some points of differentiation which it is helpful for us all to remember. Generally, a case of colic in which the temperature and visible mucous membranes are fairly normal, the pulse no more accelerated than would be expected in an excited animal and not changed much in character, bowel sounds readily audible on auscultation over the right flank, and pain clearly spasmodic, is one of simple functional disturbance which will subside, whether treated or not, within 15 hours.

With a weak pulse, cold ears, an anxious expression, a swaying gait, and not acute pain, expect a ruptured stomach. Excessive flatulence at the flank with acute distress, which is not wholly relieved by intestinal puncture, indicates either twist of the colon or an intestinal œdema due to a food allergy. These cases are fatal as a rule. This is a type of colic which is only met with definitely in certain localities in horses or mules eating certain foods such as badly cured green lucerne "hay."

The use of the stomach tube is a help in diagnosis, and although a consideration of gastric lavage properly comes under treatment its diagnostic use will be mentioned here. It is my practice as a routine measure in all cases of colic, whenever practicable, to pass the tube and pump in two or more gallons of hypertonic saline solution. In an ordinary case of simple spasmodic colic the horse takes little notice of the injection. The unimpeded passage of the solution through the intestinal canal may be evident from the subsequent loud bowel sounds on auscultating the right flank. If these sounds can be heard plainly after using the solution the owner may be told that probably his horse will recover. If, however, there is a ruptured stomach, or occlusion of the small intestine, acute distress is observed after about half of the solution has been pumped in. The injection is then stopped. If there is an impaction near the stomach, or tympany of the stomach, ingesta is apt to be discharged from the tube. In my experience either of these evidences denotes definitely a fatal ending.

In no text-book or article that I have seen is it stated that one cannot easily relieve tympany of the stomach by means of the stomach tube, but that has been my experience.

In the *Veterinary Record* of September 5, 1942, for instance, in a review of post-mortem statistics of equine colic compiled in Berlin, it is stated concerning the statistics of ruptured stomach that: "The incidence of rupture is lower than recorded by other authors due to early emptying of the stomach tube." My experience, however, is that the emptying of an engorged equine stomach by means of the stomach tube at our disposal is impracticable. This seems to be due to the fact that tympany never occurs except in an engorged stomach and that in such a condition the tube enters the stomach below the level of the semi-solid contents. Theoretically one ought to be able to go on pumping in

saline and syphoning it off again until all the solids are removed and the gases are released, but try it! I have, but in vain. Perhaps I lacked the patience, but I doubt if the procedure is practicable in a really engorged stomach. There is too much of it. One only seems to be able to get back part of the fluid one pumps in, and the tube keeps getting clogged with solids.

Moreover, the cause of the gastric engorgement must be considered. In my experience this is usually a strangulation or other block of the small intestine which is in no way affected by stomach lavage. The unfortunate horse cannot complain, but an intelligent owner might resent his doomed animal being unnecessarily worked upon for an hour or more for purposes of stomach lavage when the autopsy discloses a volvulus of the small intestine.

Treatment

Those who are still sanguine about the effect of drug treatment in colic should study the above-mentioned post-mortem statistics in the *Veterinary Record* abstract of the paper by J. Dobberstein and W. Dintner (1941) on work at the Berlin Veterinary College. In 556 deaths the P.M. examination disclosed a definite material cause in every instance, and in none, perhaps, except a few impactions was cure by means of drugs likely to be possible. Certainly there was no sign of death from simple functional colic, thus proving my statement that such cases almost always recover whether treated or not.

As regards the possibility of relieving a potentially fatal impaction, I have never observed on P.M. examination a case of equine impaction that suggested even a possibility of cure. One always saw a massive hard-packed mass of ingesta jammed into the stomach or intestine in such a way that restoration by means of a saving peristalsis was unthinkable. One had always the impression that the process of impaction had been proceeding for a considerable time and that the fatal colic was just the end of the business, wherein any attempt at treatment would be too late.

However, an English practitioner, whose name I am ashamed to say I have forgotten, recorded in the *Veterinary Record* a few years ago his belief that he had successfully treated otherwise fatal cases of impaction of the small intestine by means of acetyl choline. I doubted his conclusions at the time (my objection was also published in the *Veterinary Record*), because he made it appear that a fairly long series of his cases were all those of impaction of the small intestine, and I doubt it more now after studying the Berlin statistics. These statistics place the incidence of small intestine impaction as a cause of fatal colic at 5 per cent., or one in twenty cases.

I believe Gratzl has not published anything to show that acetylcholine is any good. Gratzl concluded that, with the possible exceptions of atropine and gastric lavage, nothing promised much in the treatment of colic. He suggested lentin, of which I have had no experience. He objected to arecoline on the rational grounds that in colic there was already an excessive excitation of the parasympathetic nervous system and arecoline increased the excitation. He also blamed arecoline in his experiments for increasing the tendency to ruptured stomach.

My own objection to arecoline is a humanitarian one, and a rational one in the absence of any real proof of its remedial value in the treatment of

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spasmodic colic. I can imagine few forms of torture more terrific than injecting large doses of arecoline into a horse with volvulus, as is done every day in veterinary practices where arecoline is used as a routine measure.

I have not made systematic experiments with proper controls, as Gratzl may have done, but I have carefully observed for many years a large number of different sorts of treatment, and my conclusions are about the same as those of Gratzl. In my experience identical results, or at least results in which there was no manifest difference, were obtained in the treatment of colic on a great scale by the following various means—except that I believe that the use of certain drastic alkaloids unnecessarily increased the pain in fatal cases :—

(1) *The British Army System:* This system—tried out on a larger number of cases of colic under systematic professional observation than almost any other —consisted in giving two to four capsules, each containing two drachms of chloral hydrate, along with a four drachm aloes physic ball. If the pain persisted two more capsules of chloral hydrate were given in an hour or two.

(2) A typical London contract horse practice used, for over fifty years, a routine drench composed of one and a half ounces of oil of turpentine and half a drachm of oil of origanum in ten ounces of raw linseed oil.

(3) English, Scottish and Irish provincial veterinary practices use a great variety of colic medicines or drenches. Some practitioners swore by sedative mixtures containing opium, sulphuric ether, chloral hydrate, and even cannabis indica. Others swore by so-called stimulant treatment based on ammonium carbonate, aloes and nux vomica.

(4) About 1907 the use of drastic alkaloidal intestinal " stimulants " such as physostigmine, eserine, and later—arecoline—became popular.

Not once in all my forty years of practice has any worthwhile evidence been brought to prove that one of these systems of medication is more likely to cure than another. All I am absolutely sure about from P.M. and other experience is that bulky linseed oil and turpentine drenches in ignorant hands killed many a horse from traumatic pneumonia and that the alkaloids can be blamed for a lot of needless torture. I wish to be quite fair to arecoline, which, I know, is highly regarded by a great number of my contemporaries.

There comes a stage in every case of spasmodic colic or slight impaction when the nerve storm is abating and recovery is at hand. At such a time the jolt that is given by arecoline undoubtedly seems to hasten resolution. I have observed this. But I have yet to see the really critical case in which it could be claimed fairly that arecoline saved the animal's life and—bearing in mind all post-mortem evidence—I cannot see how such a claim could be substantiated. Could a grain or two of arecoline, accentuating the already disorganised and ineffectual spasmodic action beating in vain against an impacted mass, safely remove the obstruction?

I do not believe it. If anything could work such a miracle it, much more likely, would be a large amount of hypertonic salt solution pumped in via the stomach tube and doing its work mechanically, irrespective of spasm. But I doubt even that. I have never been able to satisfy myself that the saline solution would relieve anything more than a non-fatal and not too massive fæcal obstruction in the large intestine. Try to liquefy and remove by means of a small

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stream of water the accumulated ingesta in a post-mortem specimen of a heavily impacted stomach or intestine and see how difficult it is.

It may be a natural scepticism that makes me thus unable to give credit to drugs in the treatment of colic, but I do not think it is only that. How, for instance, can I think highly of arecoline when the immense experiences of British Army practice, based on a drug having exactly the opposite action—chloral hydrate—gave general satisfaction? Conversely, how can I assume that chloral hydrate is reliable when practitioners using nothing but arecoline are also perfectly satisfied? If drugs had any real remedial value I think that there must have been a time in the long hours of watching when some definite sign would have been given to the clinician that here was a difference—here was something that really cured. From such a point it would have been a short step to a controlled experiment and absolute proof, but, in fact, there has never been such a point.

The only means of which I think good might be said is that persistent saline lavage does seem to help in curable impaction of the large intestine, and that atropine with morphine does definitely allay for a while the sharpest spasms of spasmodic colic. Also that chloral hydrate in large doses similarly dulls the pain of acute colic without ever doing any harm that can be proved on post-mortem examination.

It is unfortunate from a humanitarian point of view that morphine alone seems to have little anodyne or antispasmodic action in safe dosage in the treatment of equine colic. I know from personal experience what a blessed relief quite a small dose of morphine can give in human colic, but I have never observed a similar result in the horse with anything but atropine with morphine or chloral hydrate.

Discussion

Now for a rational short study of the problem. One begins by imagining what is taking place in a case of acute spasmodic colic within the abdominal cavity. The intensely painful periodic spasms show that a tremendous natural effort is being made to overcome an obstruction to the rising peristaltic waves—an obstruction which may be either traumatic or structural or functional.

As in the mechanism of parturition, the pause or rest between the spasms is obviously the means whereby energy is conserved for a periodic maximum effort.

In any natural counter-action to a pathogenic influence the full potential is exerted and there is—generally speaking—nothing useful or safe that we can do by way of stimulation to increase that potential in the acute phase of the condition. Consequently it seems to me that in acute colic one cannot do good and may do harm by trying to increase the expulsive force of a nervous and muscular paroxysm which already, in a disorganised panicky sort of way, is doing its agonising utmost.

These reflections seem to rule out drugs like arecoline altogether, and I am unable to see how mixtures of turpentine and oil, small doses of opium, ether and the like can remedially affect a violent nerve storm in the parasympathetic nervous system.

Aloes obviously must act too late to have any decisively curative effect on

a condition which is usually resolved either by death or recovery in a few hours. The value of aloes is strictly limited to cases of constipation. All that atropine or chloral hydrate or any other narcotic can do is to reduce pain without materially hindering resolution in functional colic.

As cure of any sort of colic depends upon removal or cessation of the cause it is obvious that no narcotic can "cure" a case of colic. This must be done by natural means, but the narcotic can make the process more tolerable. Consequently narcotics should be used when the pain is acute. In the beginning one had great hopes for gastric lavage by means of the stomach tube and hypertonic saline solution. It seemed rational to suppose that there would ensue a sweeping away of any irritating chemical product of indigestion or of decomposition in the food which might be the cause of the colic.

So far as acute spasmodic colic is concerned, I do not think that gastric lavage has fulfilled the hopes that were aroused when it was first practised. But in the treatment of intestinal obstruction with food or sand in removable quantities I am satisfied that gastric lavage must, and in fact does, exert a good effect. I have had several complete recoveries in cases of large bowel impaction —in which no fæces were passed for more than two days—with no other treatment than daily stomach lavage.

The increased bowel sounds following the use of gastric lavage in curable cases of impaction suggest that beneficial action is being exerted.

Use of Stomach Tube

I have had some trouble with the rather too flexible tube used in this country, but so far I have not failed to pass the tube in time by exercising patience, keeping the tube hard on the floor of the nostril, and working it backwards and forwards with the tip in the pharynx until the animal swallows and I can feel it entering the gullet. To facilitate this my tube is marked with ink at a point which is just outside the nostril when the tip is in the pharynx. A stiffer tube would be better in tending less to bend down into the larynx, but it should be noted that the lumen must not be sacrificed to rigidity if any washing out of the stomach is contemplated. The lumen must be large enough for food particles to be evacuated. I obtained from England the much stiffer and larger tube used for gastric lavage in that country, but find it too large in outside diameter for the smaller Tasmanian horses. It causes too much discomfort and hæmorrhage.

In my experience the best technique for passing the tube is to stand on the near side of the horse while the owner on the off side steadies the head by means of his hand on the bony part of the nose. Then work the tube—previously wetted—into the left nostril, going along the floor of the nostril until the pharynx is reached. Entrance to the œsophagus is apparent by the resistance to the tube and the fact that it blows back when you blow into it. With experience the feel of the tube is enough. Once in the gullet I push the tube on until the gurgitating sounds are heard when the stomach is reached. Usually these sounds can be heard by listening at the distal end of the tube. They are helpful for diagnostic purposes, being a good sign.

I am still waiting for the day when there will be a terrific rush of gas, as

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from an opened tyre valve, to signify the automatic release of gastric tympany. It has never happened in my experience and I am beginning to fear that it never will. Once the tube is in the stomach I start pumping in a bucketful of lukewarm water in which half a pound of common salt has been dissolved.

Before the bucket is empty I already know—for reasons already stated—a good deal more about the nature of the colic, and once it is empty without the appearance of any considerable distress I still hope that some good may have been done.

A STUDY OF 97 CONSECUTIVE CASES OF BOVINE DYSTOCIA AS THEY WERE ENCOUNTERED IN THE COURSE OF GENERAL PRACTICE—II

By J. B. TUTT,
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(*Concluded*)

13 Guernsey primiparous. Lay interference had resulted in severe laceration of the posterior vagina and anterior vagina and uterus with extensive bruising. Anterior presentation in the dorsal position with bilateral impacted carpal flexion. Delivered after correction but uterus prolapsed in 12 hours and was destroyed. Slaughtered.

14 Shorthorn multiparous. Twins. (1) Posterior presentation in the dorsal position with non-impacted bilateral hock flexion. (2) Anterior presentation in the dorsal position with bilateral carpal flexion. Manipulation. Recovery.

15 Shorthorn multiparous. Posterior presentation in the dorsal position with extreme impacted bilateral hip flexion. Epidural anæsthesia with total embryotomy. Recovery.

16 Guernsey multiparous. Anterior presentation on the dorsal position with left carpal flexion, head deviated to left with slight axial rotation. Manipulation. Recovery.

17 Guernsey cow. Twins. (1) Posterior presentation in the right dorso oblique position with bilateral stifle extension. (2) Anterior presentation in the ventral position, slight deviation of the head to the right and slight bilateral carpal flexion. Manipulation. Recovery.

18 Friesian heifer. Anterior presentation in the dorsal position with slight left elbow flexion. Absence of fœtal fluids and vagina very small posteriorly. Forced traction. Death.

19 Guernsey cow. Posterior presentation in the dorsal position with bilateral hock flexion posture, slight impaction. Fœtus a Schistocormus. Manipulation. Recovery.

20 Shorthorn cow. Anterior presentation in the dorsal position with full carpal extension posture. Extreme head deviation to left with axial rotation. Manipulation. Recovery.

21 Shorthorn cow. Anterior presentation in the dorsal position with slight bilateral carpal flexion. Extreme head deviation to right. Marked straining. Epidural anæsthesia and manipulation. Recovery.

22 Shorthorn cow. Twins. Anterior presentations with both heads presented with three forelegs. Epidural anæsthesia and manipulation. Recovery.

23 Shorthorn cow. Posterior presentation in the dorsal position with non-impacted bilateral hock flexion posture. Left foreleg foot nape position with spinal curvature with shortened and twisted tail. Manipulation, slight vaginal tearing. Recovery.

24 Guernsey cow. In labour five days. Decomposing seventh month fœtus

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in the left cornua. Posterior presentation in the dorsal position. Absence of fœtal fluids and uterus contracted around fœtus. T 105. Epidural anæsthesia, embryotomy and oil. Death in three hours.

25 Shorthorn heifer. Normal posterior presentation in the dorsal position. Fœtus large and impacted at the hips. Manipulation, live calf, slight vaginal tearing. Recovery.

26 Shorthorn heifer. Normal anterior presentation. A ring-like fold in the posterior vagina just anterior to the meatus urinarious, only admitting two fingers. Slaughter advised as animal in beef condition.

27 Guernsey heifer. Marked straining for six hours. Cervix not open, and did not dilate until 48 hours later, when a live calf was born unassisted in the anterior position. Recovery.

28 Guernsey cow. Anterior presentation in the right dorso lateral position with legs fully extended, head deviated to right with slight axial rotation. Manipulation. Recovery. Live calf.

29 Ayrshire cow. Hypocalcæmia. Cervix dilated and membranes intact, no straining. Anterior presentation in the right dorso lateral position, head deviated to left with slight axial rotation. Slight carpal flexion right leg. Manipulation and calcium borogluconate. Recovery. Live calf.

30 Jersey heifer. First seen straining with cervix only partially dilated. Left for 24 hours and cervix not fully open. Anterior presentation in the dorsal position with head deviation to right with slight axial rotation. Premature dead calf. Recovery.

31 Shorthorn heifer. Anterior presentation in the dorsal position but the fœtus had passed through a tear in the vaginal roof and lay between vagina and rectum in a normal anterior presentation. Epidural anæsthesia and manipulation. Dead calf. Slaughtered two days later. No lay interference.

32 Guernsey heifer. Lay interference. Anterior presentation in the dorsal position with marked head deviation to left. Vaginal laceration and bruising with marked swelling. Case hopeless. Embryotomy. Death in three days.

33 Kerry Cross cow. Anterior presentation in the dorsal position. Head deviated to the left. No fœtal fluids and womb felt tubercular. Fœtus dead and putrifying. Manipulation. Death.

34 Shorthorn cow. Twins. One delivered normally. The second a ventro transverse presentation, head to right, both hind legs and head presented. Epidural anæsthesia with amputation of the head and hind leg traction. Recovery.

35 Guernsey heifer. Posterior presentation in the dorsal position, engaged hock flexion posture. Manipulation, dead fœtus. Recovery.

36 Guernsey Cross cow. Anterior presentation in the dorsal position with slight elbow flexion. Fœtus large. Manipulation, dead fœtus. Recovery.

37 Shorthorn cow. Twins. (1) Anterior presentation in the dorsal position with left carpal flexion. (2) Normal anterior presentation. Manipulation. Live calves opposite sexes. Recovery.

38 Shorthorn cow. Anterior presentation in the right lateral position with slight head deviation on the left. Manipulation. Live calf. Prolapsed uterus three hours later and died.

39 Shorthorn cow. Twins. Two heads and three legs presented. (1) Anterior presentation in the dorsal position with right shoulder flexion and left leg fully extended. (2) Anterior presentation in the dorsal position with both forelegs fully extended. Manipulation. Live male calves. Recovery.

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40 Shorthorn cow. Anterior presentation in the normal position. Circular thickening in the vagina allowing the passage of two fingers only. This was incised and live calf delivered. Death from sepsis in four days.

41 Shorthorn cow. Posterior presentation in the dorsal position in breech position. Violent straining. Epidural anæsthesia but rupture of the uterus occurred with profuse hæmorrhage. Slaughtered.

42 Jersey heifer. Cervix not widely dilated. Anterior presentation in the dorsal position with head deviation to left with slight axial rotation. Manipulation. Dead calf. Recovery.

43 Shorthorn cow. Posterior presentation in the dorsal position with impacted bilateral hock flexion. Epidural anæsthesia. Manipulation. Dead calf. Recovery.

44 Shorthorn cow. Posterior presentation in the dorsal position with right hock flexion posture. Manipulation. Dead calf. Recovery.

45 Guernsey cow. Uterus twisted to right, corrected after throwing and rolling. Anterior presentation in the dorsal position with bilateral carpal flexion. Fœtus large. Epidural anæsthesia and total embryotomy. Recovery.

46 Shorthorn heifer. Marked twisted uterus to left. Irreducible. Slaughtered.

47 Red Poll heifer. Anterior presentation in the dorsal position with right carpal flexion and head flexion to right. Absence of fœtal fluids. Epidural anæsthesia and amputation of head. Recovery.

48 Red Poll heifer. Posterior presentation in the slight right dorso lateral position with engaged bilateral hock flexion posture. Manipulation. Recovery.

49 Shorthorn heifer. Anterior presentation in the dorsal position with right elbow flexion. Generalised emphysema of the fœtus. Vaginal constriction at the hymenal region. Manipulation with forced traction. Recovery.

50 Shorthorn heifer. Twins. (1) Posterior presentation in the right dorso lateral position with non-impacted hock flexion. (2) Similar position in the left cornua. Manipulation. Live opposite sexes of calves. Recovery.

51 Guernsey cow. Anterior presentation in the dorsal position, right leg carpal flexion, axial rotation of head. Fibrous band running from roof of vagina at cervix to lower left vaginal wall and very mobile. Manipulation. Dead calf. Recovery.

52 Shorthorn cow. Anterior presentation in the dorsal position, slight axial rotation of head with elbow flexion posture. Manipulation. Recovery.

53 Shorthorn cow. Calved one live calf 24 hours previous. (2) Posterior presentation in the dorsal position with right hip flexion and left hock flexion posture. Fœtus œdematous and putrid and uterus distended with gas. Epidural anæsthesia and amputation of left leg with forced traction. Recovery.

54 Shorthorn cow. Anterior presentation in the right dorso lateral position with head flexion to right. Epidural anæsthesia with total embryotomy. Recovery.

55 Guernsey heifer. Posterior presentation in the dorsal position with partial hock flexion and crossing of hind legs, non-impacted. Manipulation. Dead fœtus. Recovery.

56 Shorthorn heifer. Twisted uterus to right, corrected by rolling, but cervix not dilated. Four days later normal anterior presentation. Large fœtus. Forced traction. Recovery.

57 Red Poll heifer. Putrifying premature fœtus enclosed in membranes lying in vagina. Cervix closed. Removed. Recovery.

58 Shorthorn heifer. Lay interference. Anterior presentation in the dorsal

position with head flexion to right. Manipulation. Live calf. Recovery.

59 Jersey-Guernsey Cross cow. Anterior presentation in the dorsal position with bilateral elbow flexion posture. Manipulated. Live calf. Recovery.

60 Guernsey heifer. Anterior presentation in the dorsal position with bilateral elbow flexion posture. Head deviated to left. Manipulation. Live calf. Recovery.

61 Guernsey heifer. Posterior presentation in the dorsal position with bilateral hock flexion non-impacted. Pelvic inlet narrow and fœtus very short in the back. Manipulation. Recovery.

62 Shorthorn cow. Anterior presentation in the dorsal position with bilateral elbow flexion. Fœtus anasarcous and œdema of fœtal membranes. Forced traction. Recovery.

63 Shorthorn heifer. Anterior presentation in the dorsal position with bilateral carpal flexion posture with slight axial rotation of head to right. Manipulation. Dead calf. Recovery.

64 Red Poll heifer. Anterior presentation in the dorsal position with head exposed at vulva and bilateral shoulder flexion impacted. Amputation of head. Repulsion. Manipulation. Recovery.

65 Shorthorn cow. Anterior presentation in the dorsal position with slight bilateral carpal flexion and axial rotation of head to left. Manipulation. Recovery. Live calf.

66 Shorthorn heifer. Anterior presentation in the dorsal position with both forelegs exposed at vulva. Head deviated to right with slight axial rotation. Epidural anæsthesia with amputation of one leg. Recovery.

67 Guernsey cow. Two months overdue. Mummified fœtus presented in the posterior presentation in the dorsal position with bilateral hock flexion posture. Manipulation. Recovery.

68 Shorthorn cow. Premature case. Anterior presentation in the dorsal position, head deviated to right with axial rotation. Manipulation. Recovery. Dead calf.

69 Shorthorn cow. Anterior presentation in the dorsal position with slight elbow flexion posture. Head deviated to right. Manipulation. Recovery. Dead calf.

70 Guernsey cow. Anterior presentation in the dorsal position with rotation of the anterior portion of the trunk to the left lateral position with slight axial rotation of head. Manipulation. Dead calf. Recovery.

71 Shorthorn cow. Calved a dead calf 48 hours previous. (2) Anterior presentation in the dorsal position with bilateral carpal flexion with extreme extension on the hind limbs also presented. Putrifaction of fœtus. Embryotomy. Recovery.

72 Shorthorn heifer. Six months overdue. Mummified fœtus in membranes presented through partially dilated cervix. Removed. Recovery.

73 Shorthorn heifer. Premature. Anterior presentation in the dorsal position with bilateral carpal flexion, hind legs in dog sitting position. Fœtus putrified. Manipulation. Recovery.

74 Guernsey heifer. Anterior presentation in the dorsal position, head exposed at vulva with impacted left carpal and elbow flexion posture. Right leg extended. Fœtus large. Amputation of head, repulsion and manipulation. Some temporary paraplegia following delivery. Recovery.

75 Shorthorn cow. Anterior presentation on the dorsal position with impacted elbow flexion posture. Manipulation. Recovery.

76 Friesian cow. Posterior presentation in the dorsal position with calf exposed as far as the iliac tuberosities. Gross oversize of the fœtus. Animal unable to stand and lacerated as the result of lay interference. Total embryotomy but unable to remove forequarters. Slaughtered.

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77 Shorthorn cow. Posterior presentation in the dorsal position in breech posture. Epidural anæsthesia. Manipulation. Recovery. Live calf.

78 Guernsey cow. Posterior presentation in the dorsal position with engaged bilateral hock flexion posture. Manipulation. Dead calf. Recovery.

79 Shorthorn heifer. Anterior presentation in the dorsal position with left foreleg extended. Right foreleg elbow flexion posture. Marked deviation of head to right. Fœtus anasarcous. Embryotomy under epidural anæsthesia of L.F.L. and head and neck. Recovery.

80 Shorthorn heifer. Anterior presentation in the dorsal position with head deviated to left with marked axial rotation. Embryotomy of head and delivery. Recovery.

81 Shorthorn cow. Posterior presentation in the dorsal position with engaged hock flexion posture. Right metacarpus twisted under left. Manipulation. Recovery.

82 Shorthorn cow. Premature posterior presentation in the dorsal position with engaged hock flexion posture. Manipulation. Recovery.

83 Guernsey cow. Premature. Anterior presentation in the dorsal position with bilateral carpal flexion posture. Manipulation. Recovery.

84 Shorthorn heifer. Posterior presentation in the dorsal position with engaged bilateral hock flexion posture. Manipulation. Live calf. Recovery.

85 Shorthorn heifer. Schistosomus reflexus presented with the exposed bowel. Division of the trunk by embryotomy. Delivery. Recovery.

86 Shorthorn cow. Twins. (1) Posterior presentation in the dorsal position with breech posture. (2) Anterior presentation in the dorsal position with right carpal flexion posture. Epidural anæsthesia and manipulation. Dead calves. Recovery.

87 Guernsey heifer. Anterior presentation in the ventral position with head and forelegs fully extended. Vagina and vulva very small and uterus contracted around calf. Advise slaughter or Cæsarian. Slaughtered.

88 Ayrshire cow. Anterior presentation in the dorsal position with bilateral elbow flexion posture with head rotated to left. Live calf. Perineal rupture sutured and healed by first intention. Recovery.

89 Guernsey cow. Anterior presentation in the ventro transverse position with bilateral carpal flexion. Dead calf. Recovery.

90 Shorthorn heifer. Anterior presentation in the dorsal position. Vagina very narrow and thickened hymenal ring, these parts being poorly developed. Forced traction with laceration of the vaginal walls. Recovery.

91 Ayrshire cow. Anterior presentation in the dorsal position with slight elbow flexion and slight axial rotation of the head. Gross oversize of the fœtus. Evisceration of fœtus and traction and slight laceration of the vagina. Recovery.

92 Shorthorn heifer. In labour some hours with marked lay interference. Gross oversize of the fœtus. Normal anterior presentation. Uterus torn and vagina lacerated. Slaughtered.

93 Welsh Black heifer. Premature calving at eight months. Herd riddled with Brucellosis and T.B. Anterior presentation in the left lateral position. Head vertex position. Slight twist of uterus to right. Vagina small. Epidural anæsthesia. Amputation of head and forced traction. Slight vaginal laceration. Recovery.

94 Shorthorn cow. Breech presentation in the dorsal position. Anchylosis of both hip and hock joints in extended position. Epidural anæsthesia amputation of right hind leg and delivery with anal hook. Recovery.

95 Guernsey heifer. Anterior presentation in the right dorso lateral position. Head deviated to right with slight axial rotation. Manipulation. Dead calf. Recovery.

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96 Shorthorn heifer. Labour five hours. Anterior presentation in the dorsal position with head deviated to right. Gross oversize of the fœtus. Absence of fœtal fluids. Heifer fat and vaginal passage narrow. Epidural anæsthesia, with forced traction after correction. Slight vaginal tearing. Recovery.

97 Shorthorn cow. Impacted breech presentation. Epidural anæsthesia and correction after manipulation. Live calf. Recovery.

THE EFFECT OF BLAST ON THE DIAPHRAGM OF THE HORSE

By H. E. BYWATER, M.R.C.V.S.,
West Ham.

A FLYING bomb exploded in the vicinity of a stable, killing three horses. The stable was of light, semi-permanent structure and collapsed entirely when the bomb exploded (the edge of the crater being three feet from the building).

The stable housed seven horses, all of which were trapped under the debris, three being killed apparently instantaneously. Two were subsequently destroyed, one due to fracture of the mandible and nasal bones and the other due to fracture of the fetlock.

The three horses which were killed as a direct result of the blast, in addition to various other injuries, all had rupture of the diaphragm, the muscular portion of the diaphragm being lacerated in its entire length and presenting a fringe-like appearance. The lungs appeared normal except for a very few areas of congestion of about the size of a shilling piece; petechial hæmorrhages, such as are said to occur in the human subject, being absent.

PASTEURELLOSIS IN POULTRY AND WILD BIRDS IN TANGANYIKA TERRITORY

By F. G. WADDINGTON, M.R.C.V.S., B.V.Sc.,
Kenya Colony.

THE article by Messrs. Smith and Field, entitled "The Isolation of Pasteurella Aviseptica from a Turkey," published in the February issue of THE VETERINARY JOURNAL, Vol. 100, No. 2, prompted the writer to record these notes on his experience of avian pasteurellosis in Tanganyika Territory.

(a) Pasteurella Aviseptica Infection in Poultry

The poultry farm at the Mpwapwa headquarters of the Tanganyika Veterinary Department supports several hundred fowls. The flock contains a number of pure European breeds and a variety of "grades"; the latter being the result of crossing the imported pure-breds with the indigenous native fowls.

The birds are kept in ordinary open-topped wire-netting runs; in small breeding units and larger groups of young birds.

The main causes of loss were fowl typhoid and fowl pox, both of which were eventually controlled satisfactorily by vaccination.

History.—In January, 1940, the writer investigated the cause of occasional sudden deaths occurring among fowls which had been vaccinated against both fowl pox and fowl typhoid. The birds were dying singly, at intervals varying

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from a few days up to a week or so. No symptoms were shown, and the fowls were simply found dead. In some cases, the yellowish-green excreta staining the feathers of the cloacal region gave evidence of diarrhœa prior to death.

Autopsies.—Post-mortem examinations revealed the characteristic lesions of an acute septicæmia: congestion of organs, petechial hæmorrhages, etc. In addition to these non-specific lesions, there were present on the liver, multiple, scattered, white or yellowish-white, necrotic foci, varying in size from pin-point up to about an eighth of an inch in diameter.

These liver lesions gave rise to suspicions of fowl cholera; they were quite distinctive from the congestion and bronze discoloration which were commonly seen in cases of fowl typhoid.

Isolation and Identification of Causal Organism.—Smears made from the blood, liver and spleen, and stained with Giemsa, all showed the presence of numerous short, round-ended, rod-shaped bacteria; with well marked bi-polar staining.

Cultures were made from the liver, spleen and bone marrow of the femur on to plain nutrient agar slopes; and a growth occurred in 24 hours at 37 degrees C. On the solid medium the growth appeared as small discrete dewdrop-like colonies, which became more opaque with longer incubation. Sub-cultures from the solid medium were made into plain beef boullion and, after 24 hours at 37 degrees C., these showed a growth which settled at the bottom of the tube, indicating the non-motility of the bacterium.

Slides made from the cultures and stained by Gram showed pure cultures of short, round-ended, rod-shaped bacteria with Gram-negative bi-polar staining.

Sub-cultures of the organism were maintained in the pure state; but the bi-polar staining characteristic was less marked after two or three re-sowings.

Facilities for detailed bacteriological investigations were somewhat limited at that time, but the following bio-chemical tests were possible. Acid, but no gas, was produced in glucose and saccharose. No change occurred in litmus milk.

The foregoing bacteriological examination was by no means exhaustive; but, from the lesions seen in the dead birds and the staining and cultural characteristics of the organism isolated, it seemed reasonable to conclude that the disease was fowl cholera and that the pure cultures were of pasteurella aviseptica.

Pathogenicity.—As a confirmatory measure a healthy fowl, which had been isolated for a week previously, was inoculated with 0.5 c.c. of a 24-hour broth culture of the organism, injected into the pectoral muscles. The bird was found dead on the second day after inoculation. There was a yellowish-white patch of necrosis in the muscle tissue at the site of the inoculation. Within the abdominal cavity there were lesions of a septicæmia, together with white necrotic foci in the liver; similar to those seen in the natural cases of the disease.

Cultures were made from the bone-marrow of this inoculated bird, and again a pure growth, of short, round-ended, rod-shaped bacteria which showed Gram-negative bi-polar staining, was obtained.

Diagnosis.—On the evidence given above a final diagnosis of fowl cholera was made.

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This was the first occasion on which this disease had been confirmed in Tanganyika Territory; and the diagnosis was recorded in the Tanganyika Veterinary Department's Annual Report for 1939, though the cases were actually seen in early 1940.

Subsequent Course of the Outbreak.—Odd deaths for the next few weeks, after which the disease died out.

The writer is now in Kenya and, not having access to the Mpwapwa records, he cannot say exactly how many fowl cholera deaths took place. However, from memory, he is fairly certain that the total never exceeded a dozen, among the several hundred birds on the farm. There was certainly nothing in the nature of an epizootic.

Soon after the disease was confirmed, a dead vaccine was prepared and used on the flock; but, in view of other workers' opinions on this type of pasteurella vaccine, it is doubtful if it played much part in checking the disease.

(b) Pasteurellosis in Eagles

For over three years the writer had no further experience of avian pasteurellosis, until June, 1943, when he was doing rinderpest control duty on the Wembere steppe; a great stretch of wild, sparsely inhabited country, lying about two hundred and fifty miles to the north-west of Mpwapwa.

History.—A neighbour of the writer's was out shooting game on the Wembere early one morning and happened to come across a group of three, fully-grown, tawny eagles (*Aquila rapax rapax*) which were lying dead on the open plain. Other birds of the same species were seen flying in the vicinity.

The three dead birds were found on a spot, on a pathway leading from a river, where native fishermen had stopped to clean their catches. The fish offal, which was lying around, had evidently attracted the eagles, which (though usually feeding on small mammals, birds or reptiles caught by themselves) will occasionally eat carrion, if it is in a fairly fresh state.

It appeared, from the way in which they were lying dead, in such an exposed spot, that the eagles had become suddenly and acutely ill and had died after a very short illness. If symptoms had developed gradually the birds would, almost certainly, have made off for safer, and for their species, more natural roosting places, which were available, among tall trees and low rocky hills, only two or three miles away. Probably they had been in company with other eagles in the feeding area, when they became suddenly too ill to fly; then huddled together and died during the night.

One of the dead eagles was brought back to the writer for examination. The bird had been exposed to the morning sun and decomposition had started, but was not very advanced. It was assumed that the bird had died not earlier than the previous night.

Blood Films.—Before the carcase was opened, blood films were prepared from a wing vein and stained by Giemsa. On examination, these films showed a few putrefactive organisms; but, in addition, large numbers of short, round-ended, rod-shaped, bi-polar staining bacteria were well demonstrated. These organisms were morphologically similar to pasteurella aviseptica.

Autopsy.—The examination was then continued, and on opening the abdomen

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it was found that decomposition was advanced in the viscera. However, the liver was sufficiently preserved to show that it was spotted with white necrotic foci similar to those seen in cases of fowl cholera.

Bone-marrow Smears.—Thin smears were then made, from the bone-marrow of the femur, and stained with Giemsa's stain. These films showed numerous bi-polar, pasteurella-like, bacteria in a non-contaminated state.

Diagnosis.—The writer's station was very isolated and there were no laboratory facilities, except the microscope. It was therefore impracticable to attempt to isolate any organisms from the eagle carcase. Distance and lack of communications made it impossible, too, to send any material to the Mpwapwa laboratory for cultural examination.

However, from the evidence of a short acute illness with death rapidly supervening, the presence of bi-polar pasteurella-like bacteria in the blood and bone-marrow, and the necrotic areas on the liver, the writer felt justified in making a diagnosis of avian pasteurellosis.

The blood films and bone-marrow smears were sent to the Mpwapwa laboratory for examination, and the presence of bi-polar bacteria, morphologically similar to P. aviseptica, was confirmed.

A week or two later the remains of a stork were found on the plain in the same neighbourhood, but they were much too decomposed for examination.

It is quite possible that many other birds might have died in the same area; their carcases never being found, owing to the great open stretches of country and the many carrion feeders which abound therein. The discovery of the three eagles, in a comparatively fresh state, was therefore a rather exceptional piece of luck.

(c) Discussion

There are points of similarity between the history of the Mpwapwa outbreak of fowl cholera and that of the case in the turkey poult described in the article by Messrs. Smith and Field.

The Mpwapwa poultry farm was self-contained and no new birds had been admitted to it for a long period prior to the occurrence of the cases of fowl cholera. Pasteurellosis had never been diagnosed on the farm before, and the origin of the infection was decidedly obscure.

The possibility of infection from wild birds was considered and this seemed the most likely explanation of the outbreak, but there was no evidence to support it.

Like Messrs. Smith and Field, the writer expected that, if the infection was introduced by wild birds trespassing in the fowl runs, the disease would have been more widespread among the flock.

It was surprising, too, that, while there was little doubt that the deaths were due to fowl cholera of an acute type, nothing in the nature of an epizootic occurred and the disease soon died out. It seems unlikely that the pasteurella dead vaccine had much to do with this last feature.

The diagnosis of pasteurellosis in the eagle showed that the infection does exist among the wild birds of Tanganyika, and it then seemed more probable that the Mpwapwa infection came from such a source.

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It appears, then, that wild birds do suffer from pasteurella infection, which may be spread from them to domestic poultry; and that in the latter, pasteurellosis may occur sporadically and in an acute form without attaining epizootic proportions.

Summary

(1) The diagnosis of fowl cholera in poultry in Tanganyika Territory is described.
(2) A description of the identification of pasteurella aviseptica isolated from the affected birds is given.
(3) The inoculation of a healthy fowl with culture of the pasteurella, isolated from the original affected birds, and the subsequent development of fowl cholera in the experimental bird, is described.
(4) A description of what was, very probably, pasteurellosis in an eagle is given.
(5) The possible inter-relation between infection in wild birds and in domestic poultry is discussed.
(6) The view is expressed that the acute form of fowl cholera may occur sporadically, without the development of an epizootic.

Abstracts of Current Literature

WHAT TO KNOW ABOUT VITAMIN A IN (1) LIVESTOCK, (2) POULTRY, (3) SMALL ANIMALS*

ALL farm animals possess the power to convert carotene into vitamin A in the liver and store it there when the intake is low. As the amount stored varies with age and species, the arrival of deficiency symptoms varies when the intake is below the daily requirement. At birth, the fœtus contains but little vitamin A and receives its first quota from the colostrum, hence the importance of the mother's first milk. The diseases closely associated with vitamin A deficiency are abortion, keratitis, anasarca, respiratory infections, night blindness, slow growth, sterility and a manifest low resistance to respiratory and digestive infections.

In poultry, low egg production, stunting, rough plumage, infertility of eggs and marked susceptibility to intestinal worms are constant. A common symptom in fowls is film-covered, watery, purulent eyes and pustules of the mouth and throat.

Dogs and cats that are vitamin A deficient are highly susceptible to distemper, nephritis and enteritis, ocular infections and general unthriftiness. As viewed in farm animals, this avitaminosis is seen in the form of weak, germ-catching mucous membranes, the infections invited and the consequential lesions. The treatment is carotene-containing plants and, in their absence, the usual fish-liver oils as supplements.

* George H. Hart, Cliff. D. Carpenter and M. L. Morris, 1944. *Jl. Amer. Vet. Med. Assn., civ,* 802, 36-40.

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PHENOTHIAZINE-SALT MIXTURE AS AN ANTHELMINTIC IN SHEEP*

After the dependable vermicide action of phenothiazine was proved experimentally and in field use, the large (bulky) dose required, the insolubility of the drug and the repulsive taste imparted to feed, made its use cumbersome in large flocks of sheep, until it was discovered that mixing phenothiazine with common salt at the rate of 1 to 15, and giving the animals access to the mixture in lieu of the usual salt allowance, was proved markedly effective in expelling the more common Nematoda infecting ruminants. Britten, Cameron, Miller and Cameron, University of California, who have used this simple method extensively under a variety of conditions, report that the drenching of animals individually with solutions of nicotine and copper sulphate may be largely discarded. The authors conclude: "Phenothiazine at the rate of 1 to 15 is effective in controlling the common roundworms in sheep under California conditions." At this concentration, the mixture is non-toxic when animals are allowed free access to it over a period of 11 months.

* *The Cornell Veterinarian*, October, 1943.

SULPHAMETHAZINE TREATMENT OF CÆCAL COCCIDIOSIS†

Marked favourable response was produced in chicks with cæcal coccidiosis by the use, as drinking water, of a saturated solution of sulphamethazine. The test was conducted with three lots of 25 four-week-old White Leghorn chicks. Two lots were infected with a suspension of sporulated oöcysts of *Eimeria tenella* in the mash. One was treated with the sulphamethazine drinking water when the first blood was noted in the droppings. The third lot served as untreated controls. In infected untreated controls, the mortality was 88 per cent. in eight days, but only 24 per cent. in the treated group. There was no mortality in uninfected control birds. The weights averaged 332 gm. eight weeks after treatment started in infected birds, as compared with 396 gm. in those which were uninfected. The weights of survivors in the untreated infected group averaged only 250 gm.

† *Poultry Science*, 22, No. 6, page 459, Michigan Experiment Station.

CULTIVATION OF PASTEURELLA TULARENSIS IN LIQUID MEDIA‡

The difficulty of cultivating *Pasteurella tularensis* in liquid mediums was overcome by workers of the U.S. Public Health Service. Tamura and Gibby, of the Division of Infectious Diseases, National Institute of Health, Rocky Mountain Laboratory, made up such a medium from gelatine, casein hydrolysates, or amino acids, supplemented with certain accessory factors. Another one, simpler and more practical, is made up of the following formula:

Bacto-heart infusion (dehydrated) ...	32.0 gm.
Cystine	1.5 gm.
Dextrose	10.0 gm.
Bacto-hæmoglobin	5.0 gm.

The bacto-heart infusion is made of beef heart, 500 gm.; proteose peptone,

‡ *Public Health Reports*, January 21, 1944.

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10 gm.; and sodium chloride, 5 gm. per litre. Directions for preparation are given. Original inoculations are made from fresh cultures on cystine heart agar slants and then transferred to the liquid medium with loop or capillary pipette. Controls were used with each transfer. Strains of the organisms obtained from three sources (guinea-pigs, man and ticks) were then employed.

FLIES AS CARRIERS OF SALMONELLA PULLORUM*

Salmonella pullorum was recovered from the wings and feet of flies immediately after exposure to bait and for six hours after, and from the intestinal tract five days later. In each case cultures were made. Failure to infect chicks with relatively large amounts of culture suspensions was attributed to lowered virulence of the infective agent. In another experiment, a small number of chicks died after being exposed to flies infected by feeding on chick mash. Flies exposed to infected chicks furnished the most virulent material for this type of experiment. That flies are carriers of *Salmonella pullorum* when exposed to the infective agent was confirmed.

* *Canadian Journal of Public Health*, February, 1444.

THE SYNTHESIS OF VITAMINS IN THE RUMEN†

THE fact that vitamin B production in the rumen is a natural phenomenon does not mean that the bacteria of that reservoir can synthesise vitamins out of any kind of feed, nor does it furnish an excuse for feeding a diet low in vitamins. Much depends upon the activity of fermentation brought about by the action of the bacterial flora in a favourable medium. Alfalfa, for example, with its low fermentable carbohydrates and high vitamin B content, needs no great amount of bacterial fermentation to supply a sufficient amount of that micronutrient. However, a fermentable carbohydrate, like corn (maize), is required to synthesise riboflavin rapidly enough to supply the need of that factor, and to step up the synthesis of riboflavin in more ample amounts; the corn must be ground. By the same token, workers at the Ohio Agricultural Experiment Station showed that thiamin synthesis was increased when steers were fed ground corn in lieu of whole corn, and others have long since demonstrated the futility of attempting to supply normal amounts of riboflavin (vitamin G) in the milk of cows fed on timothy hay, a roughage low in protein and that vitamin. Biotin, nicotinic acid, vitamin K and pantothenic acid may be added to the list of agents which the rumen synthesises, provided the feed has a favourable chemical and physical balance; pH, protein, sulphates, temperature, coarseness and the reservoir itself must participate in this ***in vivo*** mechanism.

† *The North American Veterinarian*, February, 1944.

TOXICITY OF RED SQUILL RAT BAIT FOR ANIMALS‡

RED squill (*Urginea martima*) is used extensively as a raticide on farms because it has been found to be relatively harmless for farm animals: pigs, calves, rabbits, which may have access to the bait. Since the Ministry of Agriculture (England) reported that while chickens eat the poisoned bait more readily than pigs, the lethal dose is 10 to 30 times as large for rats. But this did not prove the relative toxicity in mammals, since high tolerance of poisons by fowl

‡ *Canadian Journal of Comparative Medicine*, August, 1943.

is not unusual. Experiments have shown that red squill is three times as toxic for female as for male rats. Testosterone seems to be antidotal since removing the ovaries does not change the toxicity. No importance is claimed, however, for this sidelight to the study of relative toxicity in farm animals.

The toxic dose for white rats was established at 50 to 800 mg. per kg. of body weight and at 200 mg. per kg. for pigs. It is, therefore, evident that low intake due to unpalatability is accountable for the low toxicity in farm animals. Farm animals do not eat red squill in lethal amounts.

Massive experimental doses caused pulmonary œdema, erosion of the bronchiolar epithelium, renal injury, severe damage throughout the gastrointestinal tract, and liver degeneration. Although the lesions were not consistent in all species tested for susceptibility, the lesions were essentially similar.

The scientific fact seems to be that the refusal of farm animals to eat this rat bait in toxic amounts accounts for its usefulness as a raticide.

News

A NEW VETERINARY SCHOOL

THE new veterinary training school for Africans has now been completed at Vom, Northern Nigeria, and students who have completed their basic science course are in residence. It will be open to pupils from all parts of the Colony and from other parts of British West Africa.

This important new training centre for African veterinary officers is part of Nigeria's policy of modernising the farming methods of the people. After their training the students from Vom will carry out such work as the supervision of the immunisation of cattle against rinderpest and other diseases, investigating outbreaks of disease and taking charge of animal clinics.

The new school has been built on the most modern lines. There are roomy, well-equipped laboratories, a lecture room and a spacious library and museum Nearby is a large clinic containing a pharmacy and an operating theatre, and stables and kennels for animal patients. The research laboratories and the farm are in the immediate vicinity. Careful attention has also been paid to the accommodation of the students. There are married and single quarters, and playing fields and a squash court are to be built. No fees are to be paid by the students, and each will receive a subsistence allowance of £36 a year while registered at the school. After successfully graduating from Vom, students will be admitted to the Government service.

The work of Nigeria's Veterinary Department has grown in importance since the war and will undoubtedly play a big part in the development of the Colony's dairy-farming, livestock and hide industries. The function of the Department is, at the same time, not confined to the prevention and eradication of such diseases as rinderpest, pleuro-pneumonia, blackwater and anthrax. It includes also the prevention of animal suffering. Another development in Nigeria's veterinary services is the opening of animal clinics, built by native administration, for the healing of wounds, the cure of lameness and the destruction of animals where no cure is possible.

Emphasis is placed on the importance of a second elementary education for

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young men who wish to take up the scientific and professional careers that are opening up for them in Nigeria. Educational leaders in the Colony are advised that English should be taught at an early age, so that pupils can qualify for admission to the schools offering courses of technical training.

Reviews

Two further National Veterinary Medical Association publications have recently been published :—

No. 6.—Report on Diseases of Farm Livestock. Section II : Diseases of Sheep.

No. 7.—Memorandum on Farm Buildings.

We welcome these additions to the list of N.V.M.A. publications. It is right that the Association should take steps to bring the relevant and up-to-date facts concerning such problems to the notice of the profession and the public. Indeed, it can be said that it is the body most competent to do so. In both books there is a mine of essential information which has evidently been collected with great thoroughness and care. We recommend the books to all who are interested in the farm and its livestock.

Copies can be obtained by non-members from the office of the National Veterinary Medical Association at 36, Golden Square, London, W.C.1. No. 6, 10s., and No. 7, 5s., both post free.

Diagnostic Methods in Veterinary Medicine. By Geo. F. Boddie, B.Sc.(Edin.), M.R.C.V.S., Professor of Medicine, Royal (Dick) Veterinary College, Edinburgh. Published by Oliver & Boyd, Ltd., Tweedale Court, Edinburgh, and 98, Great Russell Street, London, W.C. Price 15s. net.

An original type of book. Professor Boddie has put on record the signs and symptoms which a competent clinician can observe in the sick animal. Many clinicians are good diagnosticians, but often unfortunately they are unable to give to the inquiring student a logical explanation of how they arrive at a diagnosis. Prof. Boddie elucidates these problems, and by basing such explanations on sound anatomical, physiological and pathological grounds, makes clear and simple the recognition and interpretation of signs and symptoms. He has tackled his subject methodically. The first two chapters are devoted to a general discussion, the development of symptoms and signs, methods of examination and the preliminary general examination.

Thereafter he makes a more detailed study of the various systems, including the eye, the ear and skin.

In Chapter XIII the various allergic reactions which are of such importance to the modern veterinary surgeon are discussed in some detail. Chapter XIV deals with the collection of material for laboratory examination, Chapter XV with urine analysis, and Chapter XVI with clinical bacteriology.

In Chapter XVIII he has called in the aid of an expert, Dr. H. H. Holman, who deals most intricately with clinical hæmatology. This chapter is too profound for such a book, which is compiled for the practising clinician, for many of the

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techniques described by Dr. Holman are the *forte* of the skilled laboratory technician. The final chapter, on post-mortem technique, is good and will be of great assistance to the practitioner.

Criticism is based mainly on omissions, but bearing in mind the wide scope of the book, most of these are excusable in a first edition. On page 26 no mention is made of the fact that when the hair is pulled out easily, especially that of the mane, it is a sure sign of debility or disease. On page 49 are notes that only in exceptional cases are intestinal calculi felt; this is too sweeping a statement. In the classification of colic no mention is made of obstruction of the ileum, first described by R. Hudson.

Also dealing with intussusception in horses, a rare lesion, the most common one, i.e., cæco-colic, is not mentioned.

On page 147 it should be pointed out that tumour of the bladder is a frequent cause of blood in the urine.

No doubt these and other matters will be rectified in subsequent editions, and it is hoped that more illustrations will be included. One of the anatomical illustrations, Fig. 7, page 112, shows the trachea to be on the left of the aorta.

We welcome the book and recommend it to all who desire to become good clinicians, and we are certain that those who are already in that category will find in it much food for thought.

A Text-book of the Diseases of the Small Domestic Animals. By OSCAR VICTOR BRUMLEY, D.V.M., Dean of the College of Veterinary Medicine and Professor of Veterinary Medicine, The Ohio State University, Columbus, Ohio. Fourth edition. Thoroughly revised. Published by Baillière, Tindall & Cox, 8, Henrietta Street, Covent Garden, London. Pp. 422, 37 engravings. Price 27s. 6d. net.

IN his preface the author states that " The text includes all of the important diseases (medical and surgical) in a brief yet concise form arranged in systematic order, so that all facts can be obtained instantly."

He has certainly included all the important ones and also not a few which to all practical purposes are of no importance. An example of the latter is cholelithiasis (gall stones). In the account of this condition, which the author admits is quite rare, instructions are given as regards surgical operation. On the other hand, when dealing with dystokia, thirteen lines suffice for a description of " Forced extraction of the fœtus."

The illustrations are beautifully reproduced, but they are far too few, and some serve no useful purpose, for instance: Fig. 19, a dog contemplating her cystic calculi; and Fig. 18, which purports to show " Bilateral luxation of the patella." There may be such luxation, but a more apt title would be muscular atrophy of the lumbar and pelvic regions and hind limbs.

The book is almost of the nature of an encyclopædia, and in that way should serve a useful purpose for the student who desires to know the main facts of diagnosis and treatment without the necessity of wading through the mass of detail generally found in standard text-books.

The practising veterinary surgeon will find the book useful for quick reference.

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An Appreciation

GUY SUTTON, F.R.C.V.S.

GUY SUTTON died on June 11, 1944, after a short illness. He was taken suddenly ill with heart trouble and was removed to St. Mary's Hospital, where he died.

He was born on May 8, 1877, of well-to-do parents, but his father, having got through a considerable fortune, died when Sutton was ten years old. The widow and her three children were left very poor. His brother and sister died soon afterwards. He started work at the age of 14 in a City office. He loathed office work, and by the time he was 21 he had saved enough money to enter the London College. So he took under his own power the first step towards his ambition, which was to become a veterinary surgeon. Whilst at college he lived for some years with Prof. and Mrs. Pritchard, for whom he had a great regard, and to the end of his life he spoke of them with sincere affection. Pritchard took a great interest in him, and through him Sutton got to know all the outstanding veterinary surgeons of the day in London. With many of these men he formed enduring friendships. Pritchard was a frequenter of the National Sporting Club, and Sutton loved to relate anecdotes of the gay parties at that place. He would say with quiet humour that one of the main reasons that they took him there was to have someone at hand to drive the participants home. These experiences had no ill-effect; rather they tended to accentuate the good sporting instincts which were already to be seen in the young man.

He qualified M.R.C.V.S. in 1902 and went as assistant to Messrs. Batts, of Oxford Street. This was at a time when West End veterinary surgeons went about in silk hats, frock or morning coats, and paid their visits in a smart brougham. Batts were a renowned firm where assistants were treated in every way as gentlemen and, indeed, as one of the firm. Sutton often spoke with great regard of his first employers and of the grand team spirit which prevailed in their firm. In 1909 he obtained his Fellowship and in the same year joined Messrs. Ridler and Hobday in Kensington Church Street. Here he remained until the end. He and his partners built up a great practice.

Also in 1909 he married Gertrude Mary Wolfe, daughter of John Wolfe, jobmaster, Russell Square. It was through their mutual interest in horses that they met, and this mutual interest remained constant throughout his life. If he took an afternoon off it was almost invariably to take his wife to a horse show. It has been said truly that his two absorbing interests in life were his family and the horse.

When the Great War came both he and his partner, Hobday, made a great gesture. They left a most lucrative practice to give their services in the Army. Sutton served from 1915-1919 in Egypt and Palestine and the Ægean islands, reaching the rank of Major. On demobilisation he returned to Church Street, resuming his civil practice in partnership with F. Hobday and H. Stainton. The latter had kept the business going whilst the two senior partners were on service. He returned to a changed world; their great horse practice, being mostly of the light variety, had vanished. Sutton was never a man to despair. When the

horse door almost shut he opened the small animal one, and in due course energy and resource gained its reward. The practice flourished.

Now as to the man. He was short and dapper and had the carriage of a typical horseman. He had the most charming old-world manners and was courteous to all. He was a great family man, devoted to his wife and children. He would impress on his children that the things which mattered most were courtesy, integrity, courage and perseverance. These qualities he possessed to a marked degree himself. The great tragedy of his life was deafness, which assailed him in the latter half of his life. He never complained, but just carried on as if it did not exist. Fortunately he had a great sense of humour, and one saw him laugh his way through many troubles. There were few things in which he could not see a funny side. He was a great Londoner, with a profound knowledge of its history, its highways and by-ways, old houses, etc. He also liked to collect old books and sporting prints. To sit at his side and hear his reminiscences of the men and affairs of the golden days of the horse was a delight I shall always cherish. In his profession he was recognised as a highly competent man, one of the greatest integrity; in fact, a veterinary surgeon's "vet." Sir Fred Hobday once told me that "Guy Sutton was one of the best surgeons he had seen." Could any man wish for a higher testimonial?

In the photograph which we publish it will be noted that he is wearing a bow tie of the true Victorian ribbon type. Such ties are rarely seen nowadays; they are the mark of an epoch. In his case he inherited them from his father, and always wore them at the proper prescribed time every year. His people at the surgery would say: "Summer's here now. Mr. Sutton has his bow tie on."

I personally feel a great loss, for I will miss my chats with this fine character, who was so full of knowledge and of anecdotes of the glorious age of the horse. I can say truly that I learnt something on every visit. Sutton has gone, but his great spirit will remain a cherished memory to all who had the privilege of contact with him. When we in turn are gone his name will live in the Guy Sutton Memorial Fund which has been initiated by two of his clients, Mr. and Mrs. Chester Beatty, with a donation of one thousand pounds.

He leaves a widow, two sons and three daughters. One son, Peter, is a M.R.C.V.S.

J.McC.

Publishers' Notices

Copy of advertisements should be in the hands of the publishers—Baillière, Tindall and Cox—not later than the 1st of the month, if proof is required.

Letters for the Journal, literary contributions, reports, notices, books for review, exchanges, new instruments or materials, and all matter for publication (except advertisements) should be addressed to the Editor.

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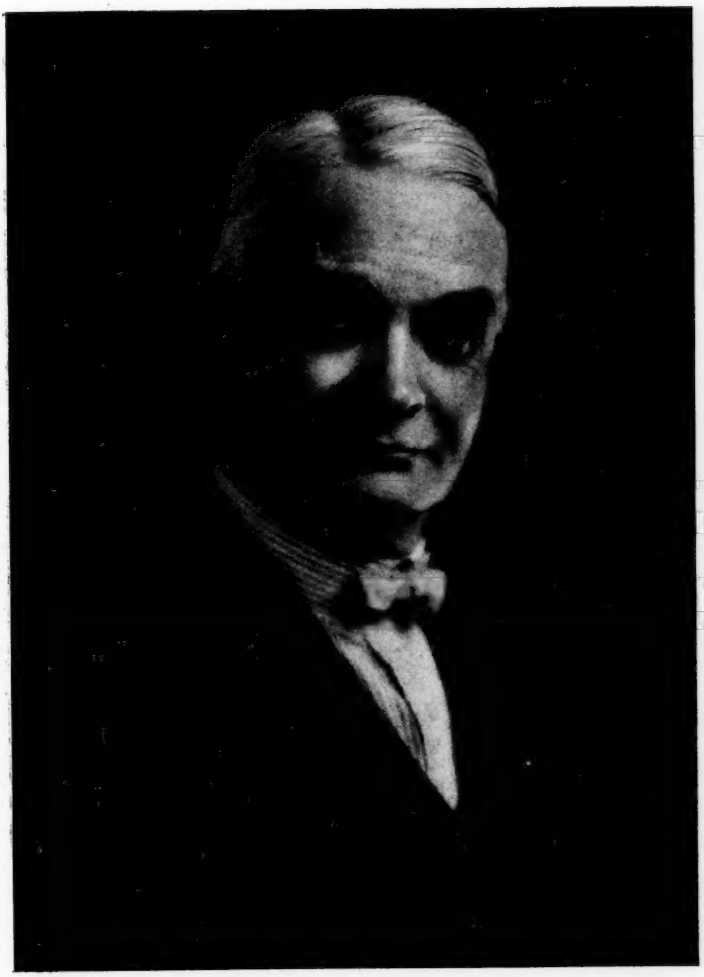

GUY SUTTON, F.R.C.V.S.

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